Social and Familial Factors in the Development of Early Childhood Asthma

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ABSTRACT. The role of social and familial factors in the development of childhood asthma by age 6 years was studied in a birth cohort of New Zealand children. Rates of asthma varied markedly with the child's sex; boys had twice the rate of asthma as girls. In addition, the factors associated with asthma varied with the child's sex. For boys, wheeze during infancy, early eczema, and parental asthma were all significant risk factors; for girls, the only risk factor was early eczema. Proportional hazards modeling of the data failed to show any significant associations between the development of asthma and a large range of other social and familial factors including breast-feeding, parental smoking habits, pets in the child's family, stress in the family, or family social background. It was concluded that asthma in early childhood appeared to be inherited to some extent, its age of expression was related to the child's sex, and it had a complex interaction with other forms of allergic disease. There was no evidence to suggest that the structure, practices, or dynamics of the child's family played a significant role in the development of asthma for children in this birth cohort. *Pediatrics* 1985;75:859–868; *childhood asthma, breast-feeding, smoking, parental asthma.*

There have been a large number of studies of the social and familial factors associated with childhood asthma. Among the factors that have been suggested to lead to increased risks of asthma are: a family history of asthma[1-10] and other atopic conditions[5,10]; a history of other atopic conditions in the child[6,7,11,12]; viral respiratory infections in early childhood[13-16]; the child's sex[4,12,13,17]; psychosocial and family stress factors[18-21]; artificial feeding[8,22-24]; parental smoking[25,26]; the presence of cats and dogs in the home[9,27]; and social background.[12,26,28-31]

However, the conclusions drawn from these studies have been limited by the fact that they have often been conducted upon small samples, from selected clinical populations, using cross-sectional or retrospective case-control designs. There appears to have been no prospective study that has examined the role of social and familial factors in the development of asthma in a large and representative population of children.

This paper reports on the results of a 6-year prospective study of the development of asthma in a birth cohort of New Zealand children. The aims of the study were: (1) to identify the social and familial factors associated with increased risks of asthma in early childhood, and (2) to develop a proportional hazards model to describe the way in which various social and familial factors and combinations of these factors influenced the likelihood of developing asthma.

METHOD

The data were collected during the first 6 years of the Christchurch Child Development Study. In this study, a birth cohort of Christchurch (New Zealand) children has been studied at birth, age 4 months, and at annual intervals to age 6 years. At each stage, information was collected on the child's health, family social background, and other factors by means of a structured interview with the child's mother supplemented by information from hospital records, general practitioner's notes, and diary of medical attendances kept by the mother. The methods of data collection and quality control have been described in detail in previous papers.[7,32-36]

From the data base of the study the following

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variables were selected for analysis: childhood asthma, family history of atopy, child health in the first year, early feeding and home environment, psychosocial factors, and family social background.

Childhood Asthma

This was based on whether the child had made two medical consultations before the age of 6 years for wheeze or associated symptoms that were diagnosed as asthma or wheezy bronchitis. Wheezy bronchitis was included in the definition of asthma on the basis of the conclusions of Williams and McNicol[36] that the two conditions were indistinguishable. (Only 8% of all diagnoses were for wheezy bronchitis.) The criterion of at least two diagnoses was used as there is some uncertainty about the significance of a single diagnosis of asthma in early childhood. The frequency distribution of the number of medical attendances for asthma is given in Table 1, which shows that by age 6 years, 10.3% of cohort members had made two or more attendances. In all cases, the diagnosis of asthma or wheezy bronchitis was made after the child was 1 year old. An analysis of the effects of varying the stringency of the definition of asthma and of excluding diagnoses of wheezy bronchitis is given under "Results." Information on childhood asthma and wheezy bronchitis was obtained from an annual diary of medical attendances kept by the child's mother in 60% of cases. In the remaining 40% of cases, this information was based on maternal recall supplemented by direct contacts with the child's family doctor.[7,35] The majority (94%) of asthmatic attacks were treated by the child's family doctor, but nonetheless 24 of the 109 (22%) children who suffered two or more asthmatic episodes had been admitted to hospital for asthma.

Family History of Atopy

Information on the history of atopic conditions (both past and present) in both of the child's biologic parents and among the child's full siblings was collected prospectively at the initial (birth) interview.[7] From this information, four dichotomous measures were constructed: (1) parental asthma—whether there was a known history of asthma in one or both parents; (2) parental eczema—whether there was a known history of eczema in one or both parents; (3) parental allergic rhinitis—whether there was a known history of allergic rhinitis in one or both parents; and (4) sibling asthma—whether there was a known history of asthma in any of the child's full siblings. For the measures of parental atopy, consideration was given to analyzing both maternal and paternal atopy separately. However, it was found that combined indices based on a history of atopy in either parent were as effective predictors as measures based on data for each parent.

TABLE 1. Frequency Distribution of Number of Medical Consultations for Asthma (Ages 0 to 6 Years)

No. of Consultations	No.	%
0	915	86.6
1	32	3.0
2	26	2.5
3	20	1.9
4	12	1.1
5–9	25	2.4
10–14	13	1.2
15*	13	1.2
Total	1,056	100.0

Child Health in First Year

The following measures were constructed on the basis of maternal reports, diary records, and records of general practitioner and hospital attendances in the first year: (1) eczema—whether the child had at least one medical attendance for skin rash that was diagnosed as eczema[33,34] (Because information on medical attendances for eczema were based on maternal reports supplemented by information from medical records, it was not possible to distinguish reliably between atopic and seborrheic eczema.); (2) wheeze—whether the child had attended a medical practitioner for wheezy chest not diagnosed as asthma, or the mother reported respiratory wheeze that did not require medical attention[37,38]; (3) lower respiratory tract infections—the total number of episodes of bronchitis, bronchiolitis, or pneumonia treated by a medical practitioner[37,38]; and (4) total respiratory infections—the total number of episodes of respiratory tract illness (either upper or lower) treated by a medical practitioner.[37]

Early Feeding and Home Environment

The following measures were used as indicators of the child's early feeding history and home environment:

Infant Milk Diet (Age 0 to 4 Months). This was based on information collected from (1) obstetric unit notes of the child's feeding history shortly after birth, (2) a diary record kept by the mother on the child's feeding history from birth to age 4 months (85% of mothers kept such a diary), and (3) questioning the mother about the child's feeding history to determine whether he or she had ever been given any cow's milk. (A detailed account of methods for assessing the breast-feeding history of this cohort has been published previously.[33–35])

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The early milk feeding history of the sample was classified as follows: (1) children who had been totally bottle fed since birth and never received any breast milk; (2) children who had been breast-fed but who had received some cow's milk before age 4 months; and (3) children whose early milk diet was breast milk only and who had never received cow's milk by age 4 months.

Parental Smoking. Each year, information was collected on the smoking habits of the child's parents. The history of parental smoking over the survey period was classified as follows: (1) neither parent smoked at any time; (2) one parent smoked at some time during the survey period; and (3) both parents smoked at some time during the study period.

Cats/Dogs in the Home. Records included information on whether the family had a pet cat or dog.

Psychosocial Factors

To measure the amount of stress, adversity, and social readjustment experienced by the family during the study period the following three measures were constructed.

Family Life Events to Age 6 Years. Each year from 2 years to 6 years, mothers were interviewed using a modified version of the Holmes and Rahe Social Readjustment Rating Scale.[39] This consisted of 20 items which covered such areas as illness in the family, bereavement, financial problems, and marital disharmony. The characteristics of the scale have been described in detail by Beautrais et al.[32] To measure the amount of adversity faced by the family, a life events score was constructed by summing the total number of life events reported over the study period.

Maternal Depression. At 5 years and 6 years, mothers were interviewed on a modified version of the Levine-Pilowsky Depression Questionnaire.[40,41] The scale consisted of 37 items measuring various symptoms of depression. The characteristics of the scale have been described by Fergusson et al.[42] To measure the mother's general level of depression, a scale score was constructed by summing the total number of depressive symptoms reported by the mother over the 2-year period.

Changes of Residence. Records included information on the number of changes of residence experienced by the child from birth to age 6 years.

Family Social Background

The following measures were used as indicators of the family's social situation: (1) maternal age at the birth of the survey child; (2) maternal education classified as—no formal qualifications, secondary qualifications (NZ School Certificate or University Entrance), or tertiary qualifications (University degree or tertiary technical diploma); (3) child's ethnic status—Maori/Pacific Island *v* European/other; (4) family socioeconomic status—based on the Elley-Irving[43] scale of socioeconomic status for New Zealand, which classifies the population into six classes on the basis of parental occupation; and (5) family size at the child's birth.

Sample Size and Response Rates

The analysis is based on a total of 1,056 children for whom complete data on all the variables in the analysis were available. This number represents 83% of the initial cohort of 1,265 children and 91% of the cohort of children who were still alive and resident in New Zealand at age 6 years. Comparison of the obtained sample with the characteristics of the initial cohort indicated that no significant biases had been introduced as a result of sample attrition.

RESULTS

Risk Factors Associated with Development of Asthma

The association between the proportions of children experiencing two or more episodes of asthma by age 6 years and a series of measures of family and social background is shown in Table 2. The results are shown separately for boys and girls, and each association is tested for statistical significance by the χ^2 test. As shown in Table 2: (1) The rate of asthma was higher among boys than girls: 14.3% of boys had developed asthma by age 6 years in contrast to only 6.3% of girls ($\chi^2 = 18.20$; $df = 1$; $P < .0001$). (2) The factors associated with increased risks of asthma differed markedly between the sexes. For girls, the only significant risk factor was eczema in the first year: girls who had eczema were five times more likely to develop asthma ($P < .0001$). For boys, there was also a significant association between asthma and early eczema ($P < .0001$). However, in contrast to girls, boys were more likely to develop asthma when there was a history of early wheeze ($P < .0001$); when there was a parental history of asthma ($P < .0001$) or allergic rhinitis ($P < .01$); and when other siblings had asthma ($P < .01$). (3) For both sexes there were no apparent associations between rates of asthma and breast-feeding, parental smoking, the presence of cats and dogs, various stresses in the family and family social background.

Because many of the variables in Table 2 were intercorrelated, the results do not indicate the net

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TABLE 2. Proportions of Children with Two or More Medical Consultations for Asthma (Ages 0 to 6 Years) by Sex of Child and Familial and Social Factors

Variable	Boys	Girls	Total
Family history of atopy			
Parental asthma			
No asthma	11.2 (48/428)	5.8 (25/433)	8.5 (73/861)
Asthma	26.9 (28/104)	8.8 (8/91)	18.5 (36/195)
Significance	$P < .0001$	NS	$P < .0001$
Parental eczema			
No eczema	12.8 (54/422)	5.8 (23/397)	9.4 (77/819)
Eczema	20.0 (22/110)	7.9 (10/127)	13.5 (32/237)
Significance	NS	NS	NS
Parental allergic rhinitis			
No allergic rhinitis	11.2 (39/347)	5.8 (21/363)	8.5 (60/710)
Allergic rhinitis	20.0 (37/185)	7.5 (12/161)	14.2 (49/346)
Significance	$P < .01$	NS	$P < .01$
Sibling asthma			
No siblings with asthma	12.8 (60/469)	6.2 (30/483)	9.5 (90/952)
Siblings with asthma	25.4 (16/63)	7.3 (3/41)	18.3 (19/104)
Significance	$P < .01$	NS	$P < .01$
Child health in first year			
Eczema			
No eczema	12.1 (59/489)	4.9 (24/488)	8.5 (83/977)
Eczema	39.5 (17/43)	25.0 (9/36)	32.9 (26/79)
Significance	$P < .0001$	$P < .0001$	$P < .0001$
Wheeze			
No wheeze	8.9 (26/293)	6.5 (21/325)	7.6 (47/618)
Wheeze	20.9 (50/239)	6.0 (12/199)	14.2 (62/438)
Significance	$P < .0001$	NS	$P < .001$
Lower respiratory tract infections			
0	13.5 (64/474)	6.2 (30/482)	9.8 (94/956)
At least 1	20.6 (12/58)	7.1 (3/42)	15.0 (15/100)
Significance	NS	NS	NS
Total respiratory tract infections			
0	14.6 (25/171)	7.7 (14/181)	11.1 (39/352)
1–2	11.8 (31/262)	5.2 (13/250)	8.6 (44/512)
3*	20.2 (20/99)	6.5 (6/93)	13.5 (26/192)
Significance	NS	NS	NS
Feeding and home environment factors			
Infant milk diet (0–4 mo)			
Bottle milk only	13.0 (16/123)	7.1 (8/113)	10.2 (24/236)
Breast milk and bottle milk	13.3 (42/315)	5.7 (18/314)	9.5 (60/629)
Breast milk only	19.1 (18/94)	7.2 (7/97)	13.1 (25/191)
Significance	NS	NS	NS
Parental smoking			
Neither parent smoked	14.6 (33/226)	7.4 (17/229)	11.0 (50/455)
One parent smoked	12.0 (21/175)	7.1 (12/169)	9.6 (33/344)
Both parents smoked	16.8 (22/131)	3.2 (4/126)	10.1 (26/257)
Significance	NS	NS	NS
Cats/dogs in home			
No	14.4 (15/104)	11.2 (10/89)	12.9 (25/193)
Yes	14.2 (61/428)	5.3 (23/435)	9.7 (84/863)
Significance	NS	NS	NS
Psychosocial factors			
Family life events (1–6 yr)			
0–4 events	12.7 (10/79)	4.7 (5/107)	8.1 (15/186)
5–9 events	13.5 (29/215)	6.6 (13/197)	10.2 (42/412)
10–14 events	15.5 (23/148)	4.8 (5/104)	11.1 (28/252)
15* events	15.6 (14/90)	8.6 (10/116)	11.7 (24/206)
Significance	NS	NS	NS
Maternal depression score (5–6 yr)			
0–4 symptoms	11.4 (29/255)	5.3 (13/244)	8.4 (42/499)
5–9 symptoms	16.8 (16/95)	8.2 (9/110)	12.2 (25/205)
10–14 symptoms	22.4 (15/67)	5.3 (3/57)	14.5 (18/124)
15–19 symptoms	18.9 (7/37)	4.8 (2/42)	11.4 (9/79)
20* symptoms	11.5 (9/78)	8.5 (6/71)	10.1 (15/149)
Significance	NS	NS	NS

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TABLE 2—*Continued*

Variable	Boys	Girls	Total
Changes of residence (0–6 yr)			
0	12.0 (22/183)	6.0 (11/182)	9.0 (33/365)
1–2	16.4 (33/201)	5.2 (10/194)	10.9 (43/395)
3–4	15.1 (13/86)	5.6 (5/89)	10.3 (18/175)
5*	12.9 (8/62)	11.9 (7/59)	12.4 (15/121)
Significance	NS	NS	NS
Family social background			
Maternal age			
<20 yr	19.1 (9/47)	4.3 (2/47)	11.7 (11/94)
20–24 yr	14.5 (25/172)	7.4 (11/148)	11.3 (36/320)
25–29 yr	15.5 (31/200)	7.2 (16/221)	11.2 (47/421)
≥30 yr	9.7 (11/113)	3.7 (4/108)	6.8 (15/221)
Significance	NS	NS	NS
Maternal education			
No formal qualifications	15.5 (42/271)	6.2 (17/273)	10.8 (59/544)
Secondary qualifications	13.3 (22/166)	6.4 (9/140)	10.1 (31/306)
Tertiary qualifications	12.6 (12/95)	6.3 (7/111)	9.2 (19/206)
Significance	NS	NS	NS
Child's ethnic status			
Maori/Pacific Island	18.5 (15/81)	3.0 (2/66)	11.6 (17/147)
European/other	13.5 (61/451)	6.8 (31/458)	10.1 (92/909)
Significance	NS	NS	NS
Socioeconomic status			
Professional, executive	14.9 (15/101)	4.3 (5/116)	9.2 (20/217)
Clerical, technical, skilled	12.8 (36/282)	6.3 (18/284)	9.5 (54/566)
Semiskilled, unskilled, unemployed	16.8 (25/149)	8.1 (10/124)	12.8 (35/273)
Significance	NS	NS	NS
Family size			
1	11.9 (25/210)	6.8 (13/190)	9.5 (38/400)
2	15.4 (28/182)	6.7 (13/195)	10.9 (41/377)
3	15.3 (15/98)	7.1 (7/99)	11.2 (22/197)
4*	19.0 (8/42)	0.0 (0/40)	9.8 (8/82)
Significance	NS	NS	NS
Total	14.3 (76/532)	6.3 (33/524)	10.3 (109/1056)

contributions of each of the factors to the rate of asthma during the 6-year period. We describe below a proportional hazards regression model designed to estimate the net effects of the predictor variables on rates of asthma for boys and girls over the period from ages 1 to 6 years.

Proportional Hazards Model of Development of Asthma

The mathematical basis of the proportional hazards model can be summarized briefly as follows. (For a complete mathematical formulation of the model the reader is referred to Cox[44] or Kalbfleisch and Prentice.[45]) Consider some population or sample observed over a series of time intervals (t) during which some subjects are observed to fail (ie, become asthmatic). The distribution of failures over time defines the survivorship function: $S_t = Pr\ (T \geq t)$, where S_t denotes the survivorship probability (Pr) to time t and T is the time to failure.

Associated with the survival function is the hazard function $\lambda(t)$. The hazard at any time t is defined as the conditional probability of failure at time t, given that failure has not occurred prior to this time. An alternative and intuitively more meaningful description of the hazard is the instantaneous failure rate.

Next, consider the situation in which subjects may be classified according to some series of variables or covariates that are assumed to influence the likelihood of failure. Let these covariates be represented by a vector of values z for each subject. The aim of the proportional hazards model is to describe the way in which the hazard varies over time with the set of covariate values. The model assumes the existence of a base-line group of subjects whose vector of covariate values is arbitrarily set to zero. It is also assumed that the effects of the covariate values are to scale the hazard over time in a way that is proportional to the hazard function for this base-line group. This model is: $\lambda(t;z) = \lambda_0(t)e^{z\beta}$, where $\lambda(t;z)$ denotes the hazard at time t for a group of subjects with covariate vector z, $\lambda_0(t)$

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is the hazard at time t for the base-line population with covariate values of 0, and β is a vector of regression-like coefficients. The parameters of the model may be estimated by maximum likelihood techniques, and estimates of the asymptotic standard errors of the coefficients β are available.[45]

To examine the net contributions of the risk factors in Table 2 to variations in rates of asthma, a stepwise proportional hazards model was fitted to the data. In this analysis, the age at which the child first developed asthma was defined as the point at which the first of at least two medical attendances for asthma or wheezy bronchitis occurred. In view of the differences seen in Table 2, separate models were fitted for boys and girls. This analysis showed that when all variables were considered, only three of these (parental asthma, early wheeze, early eczema) were significantly related to the development of asthma in boys. For girls, the only significant risk factor was early eczema. The results of the analysis are summarized in Table 3, which shows the significance of each of the risk factors in the model and the values of the proportional hazards coefficient ($e^{i\beta}$) for each level of each variable. This coefficient may be interpreted in a way that is analogous to the more familiar notion of relative risk: the increase in the instantaneous risk of asthma that is associated with a particular factor when compared with the risk for the base-line population. Definitions of the base-line populations for boys and girls are given in the footnote to Table 3. For boys, the presence of eczema increased the risk of asthma by 3.45 times over the risk for the base-line group; a parental history of asthma increased the risk by a factor of 2.78; and wheeze in the first year by a factor of 2.39. For girls, the presence of early eczema increased the risk of asthma by a factor of 5.80.

From the results of proportional hazards analysis, estimates were obtained of the risk of experiencing two or more episodes of asthma by the age of 6 years conditional on various combinations of risk factors. The results of this analysis are summarized in Table 4, which shows the estimated cumulative rate of asthma at each age conditional on the number of significant factors that the child had. For boys, three factors—early eczema, early wheeze, and parental asthma—were considered; for girls, the only factor was early eczema. The results show: (1) For boys there was considerable variation in the risk of asthma conditional on the number of significant risk factors that were present. Boys who had all three factors (early eczema, early wheeze, and parental asthma) had a probablity of approximately 80% of developing asthma by age 6 years. In contrast, those with none of these factors had a risk of only 7%. The groups of subjects with one or two of the risk factors had results that lay between these extremes. (2) For girls, variations in prognosis were less marked. However, girls who developed early eczema had rates of asthma that were slightly more than five times higher than girls who did not have early eczema.

TABLE 3. Estimated Proportional Hazards Coefficients for Levels of Significant Factors

Variable	Boys* $e^{i\beta}$	Significance	Girls* $e^{i\beta}$	Significance
Child eczema				
No eczema	1	$P < .001$	1	$P < .001$
Eczema	3.45		5.80	
Child wheeze				
No wheeze	1	$P < .001$	...	NS
Wheeze	2.39		...	
Parental asthma				
No asthma	1	$P < .001$	...	NS
Asthma	2.78		...	

* Base-line populations for the two models are: (1) for boys—children with no eczema or wheeze in the first year and without a history of parental asthma; (2) for girls—children with no eczema in the first year.

Sensitivity Analysis

Gregg[4] notes that estimates of the prevalence of asthma depend on the stringency of the criteria used to define the condition. To examine the effects of varying the stringency of the definition of asthma on both the prevalence of asthma and the factors associated with the condition, the results were reanalyzed using a series of definitions of asthma of increasing stringency. These definitions required that the child was classified as asthmatic only after he or she had suffered 3, 4, 5, or 6 episodes of wheeze medically diagnosed as asthma or wheezy bronchitis. The results of this analysis are given in Table 5, which shows for each definition the estimated rate of asthma for boys and girls and the proportional hazards coefficients for each risk factor for each analysis. The table shows: (1) The estimated rate of asthma varied sharply with vary-

TABLE 4. Estimated Cumulative Rates of Asthma (per 100 Children Aged 2 to 6 Years) by Number of Significant Risk Factors

No. of Risk Factors	2 yr	3 yr	4 yr	5 yr	6 yr
Boys					
0	1.5	3.0	4.6	5.8	6.8
1	3.7	7.4	11.1	14.0	16.1
2	10.5	20.2	29.1	35.7	40.1
3	29.6	50.9	66.2	75.1	80.1
Girls					
0	1.1	2.2	3.4	4.4	5.4
1	6.0	12.2	18.0	23.2	27.6

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TABLE 5. Proportional Hazards Coefficients (Significance of Factor) for Models Fitted to Rates of Asthma Based on Definitions of Varying Stringency

	No. of Diagnoses Before Child Was Classified as Asthmatic				
	2	3	4	5	6
Boys					
Variable: early eczema	3.45	4.04	4.36	4.18	5.52
Significance	$P < .001$	$P < .001$	$P < .001$	$P < .001$	$P < .001$
Wheeze	2.39	2.79	2.66	3.89	3.40
Significance	$P < .001$	$P < .001$	$P < .001$	$P < .001$	$P < .001$
Parental asthma	2.78	2.90	3.21	3.21	3.63
Significance	$P < .001$	$P < .001$	$P < .001$	$P < .001$	$P < .001$
Rate of asthma	14.3%	11.3%	8.6%	7.3%	6.0%
Girls					
Variable: early eczema	5.80	5.06	3.99	4.20	—*
Significance	$P < .001$	$P < .01$	$P < .05$	$P < .05$	
Rate of asthma	6.3%	4.4%	3.4%	2.5%	2.3%
Overall rate of asthma	10.3%	7.8%	5.9%	4.8%	4.2%

* The number of subjects with asthma was insufficient to estimate coefficient.

ing definitions of the condition. For the total cohort, the rate to age 6 years was 10.3% if two or more episodes was used as the basis of the classification and reduced to 4.2% if six or more episodes was used as a criterion. (2) For boys, the proportional hazards coefficients for each risk factor show a clear tendency to increase as the definition of asthma becomes more stringent. This result is to be anticipated as one would expect that as the severity of the child's asthma increased, the discriminatory power of the risk factors would increase. However, for girls, there is a decline in the values of the proportional hazards coefficients, suggesting that as the stringency of the definition of asthma increased, there was decreased predictive power. The reasons for this are not entirely clear, but it seems possible that the decline in the proportional hazards coefficients reflects the fact that very few girls suffered a significant number of asthmatic attacks, and it is possible that the small numbers studied may have influenced the stability of the proportional hazards coefficients. (3) In any event, the analysis shows that irrespective of the stringency of the definition of asthma, the same constellation of risk factors emerges as being associated with the condition, and, accordingly, it is unlikely that the conclusions drawn in this study can be ascribed to the way in which asthma was defined.

In addition, there has been some debate as to whether wheezy bronchitis and asthma are distinct conditions. To examine the effects of including or excluding diagnoses of wheezy bronchitis from the definitions of asthma, the results were re-analyzed excluding all diagnoses of wheezy bronchitis. As might be expected from the fact that only 8% of all diagnoses were for wheezy bronchitis, the results did not differ depending on whether wheezy bronchitis was included or excluded from the definition.

DISCUSSION

This longitudinal study suggests that a substantial proportion of children had been determined to be suffering from one or more episodes of asthma or wheezy bronchitis by age 6 years. Depending on the stringency of the criteria used to define the child as asthmatic, the proportion of children classified as asthmatic ranged from 10.3% (for those with two or more episodes) to 4.2% (for those with six or more episodes). Despite the fact that the prevalence and incidence of asthma varied with the stringency of definition of the condition, the risk factors associated with asthma remained invariant.

The most notable aspect of the findings was the way in which the child's sex influenced not only the risk of asthma but also the factors that were associated with the condition. Boys had more than twice the rate of asthma; this finding is in agreement with several other reports.[4,7,12,13,17] In addition, the factors associated with the development of asthma in boys differed from those associated with the development of asthma in girls. For boys, parental asthma, early eczema, and wheeze in the first year were significant risk factors. For girls, the only significant risk factor was early eczema. These trends were reflected in the predictability of the condition: it was possible to identify boys with risks of asthma as high as 80% by age 6 years and as low as 7%; by comparison, the prediction of asthma for girls was modest.

The pervasive influence of the child's sex on both the prevalence and correlates of early asthma could suggest that asthma is a sex-limited or sex-influ-

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enced condition. However, the possibility has been considered in a previous report on this cohort and a more likely explanation would seem to be that the condition is sex-expressed, with genetically susceptible boys expressing their asthmatic tendencies at the earlier age than genetically susceptible girls.[7] This hypothesis would also account for the changing sex ratios that have been observed in childhood asthma; the condition is more common in boys in early and middle childhood and equally frequent in both sexes in later childhood.[4,5]

It has been long assumed that asthma is related to some generalized tendency to atopic disease so that conditions such as asthma, eczema, and allergic rhinitis tend to run in families.[46] The findings of this and a previous study[7] suggest that this conclusion is an oversimplification inasmuch as detailed analysis of family resemblance in asthma and eczema for this cohort has suggested the presence of three quite distinct components of "inheritance": (1) an asthma-specific tendency whereby asthma in parents is associated with asthma in the child; (2) an eczema-specific tendency whereby eczema in the parents is associated with eczema in the child; and (3) a generalized atopic tendency for both asthma and eczema to occur together. These findings, coupled with the way in which asthma is influenced by the child's sex, suggest the presence of a complicated and at present poorly understood mode of inheritance which cannot be summarized by the unitary concept of atopy.

Several authors[13–16] have suggested that early viral respiratory infection may predispose children to develop asthma. The findings of our study provide only weak support for this view. Overall, there was no association between the rate of respiratory illness during early life and asthma. However, it is possible that this finding is misleading because it may be that only specific types of viral infection predispose children to develop asthma and accordingly one might not expect to find a strong association between overall rates of respiratory illness and subsequent asthma. Unfortunately, it was not possible in this study to classify respiratory illness on the basis of the source of the infection. The correlation between early wheeze and subsequent asthma might suggest a common source of viral infection which predisposes children to wheeze during early life and to develop subsequent asthma, but at the same time it is also possible that the association may simply reflect the difficulties of diagnosing asthma during infancy and that the children who were described as wheezy were merely manifesting the first stages of later asthma.

It has been held that risks of asthma and other forms of atopy can be reduced by exclusive breast-feeding.[9,22–24,47–50] However, in this and several previous studies of the cohort,[33–35] we have been unable to demonstrate benefits for breast-feeding in the reduction of atopic disease. Moreover, the view that breast-feeding prevents atopy has come under criticism recently with many studies reporting no effect or in some cases increases in atopy among breast-fed children.[33–35,51–54] It is notable that in this study, breast-fed boys had higher rates of asthma than other children, although this difference did not reach statistical significance. Although the role of breast-feeding in the prevention of asthma and other forms of atopy remains controversial, it may be fairly claimed that it is unlikely that infant feeding patterns make a major contribution to variability in the risk of asthma or eczema in the child population. However, as we have noted previously it is possible that highly selected subgroups of children may benefit from breast-feeding.[35]

It has been suggested that cigarette smoking may trigger or exacerbate attacks in patients suffering from asthma,[25] and this has led to the speculation that parental smoking may lead to the development of asthma in children.[9] However, we were unable to find any effect for parental smoking on rates of asthma and this confirms the findings of a previous longitudinal study.[8] On the other hand, Gortmaker et al[26] did find a small but statistically significant tendency for the rate of asthmatic attacks to be higher in families in which parents smoked. Collectively, these findings suggest that although parental smoking may increase the risk of asthmatic attacks, it is not implicated in the etiology or development of the condition. In previous studies of this cohort,[27,28] we have found an association between parental smoking and lower respiratory tract infection in children, and it would appear in confirmation of the conclusion of Leeder et al.[29] that although parental smoking may play a role in increasing susceptibility to lower respiratory tract infection, it does not appear to be implicated in the development of asthma in early childhood.

It has often been suggested that asthma is a psychosomatic illness triggered or caused by various social or personality factors. Recently, this view has fallen into some disrepute and there have been a large number of criticisms of the theory that asthma is a psychosomatic condition.[18,19] Our study indicates that stress in the family is not related to the development of childhood asthma and this coupled with the contradictory and confused findings on both childhood psychopathology and abnormal parenting among asthmatic children tends to support the conclusions of Werry[19] that there is no good evidence that asthma is necessarily a psychosomatic disease. In a previous study of this cohort,[32]

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we have been able to show that a series of conditions including lower respiratory tract illness, gastroenteritis, and accidents are influenced by family stressors. The fact that a similar finding does not hold for asthma casts further doubt on the alleged psychosomatic basis of the condition.

Family social background and related factors were unrelated to risks of asthma in early childhood. This result is not entirely consistent with previous findings that New Zealand Polynesian children have a greater risk of asthma than white children.[30,31] The reasons for this difference are not clear, but it is possible that it arises from the age of the children being studied or from the fact that the majority of children nominally classified as Polynesian in this study had 25% or less Polynesian ancestry.[55]

Asthma afflicts a significant minority of the child population and its etiology is poorly understood. The search for social and familial correlates has led to a large and uneven research literature, suggesting the way in which various social and environmental factors may contribute to the development of asthma. This 6-year prospective study indicates that early childhood asthma appears to be inherited to some extent, its age of expression is related to the child's sex, and it has a complex interaction with other forms of allergic disease.[7] It is not related to infant feeding practices, smoking in the family, the presence of pets in the house, stress in the family, or the family's general social or economic situation. This is not to say that in individual cases these factors may not play a role in the development and onset of asthma, but rather that their role in contributing to the overall variations in the rate of the condition in the general child population (at least during early childhood) appears to be negligible. This would suggest that the etiologic basis of asthma is more likely to be found in studies of the genetic, physiologic, and immunologic basis of the condition rather than through an examination of the structure, practices, or dynamics of the family of the asthmatic child.

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ARE INTENSIVE CARE UNITS FUTILE?

[An] expert who worked for five years at a hospital in Cali, Colombia, told how the low-weight newborn mortality rate dropped greatly after an intensive care unit with all of the necessary equipment and trained personnel was opened at his hospital.

"But then we did a follow-up study to see what happened after the babies went home," he said "and we discovered that 75% of the infants were dead in six months from infections and malnutrition."

Submitted by Student

From Nelson H: Colombians 'pack' infants to mothers. *LA Times*, April 3, 1984.

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